

WEST Search History

DATE: Monday, May 13, 2002

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR</i>			
L4	L3 and wilms	1	L4
L3	hla-A2.1 or hla-a0201	135	L3
L2	L1 and wilms	10	L2
L1	(stauss)[IN] OR (gao)[IN]	3542	L1

END OF SEARCH HISTORY

8

(FILE 'HOME' ENTERED AT 10:25:06 ON 13 MAY 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 10:25:20 ON 13 MAY 2002

L1 2814 S STAUSS H?/AU OR GAO L?/AU
L2 3 S L1 AND (GATA OR WT-1)
L3 3 S L1 AND (GATA-1 OR WT-1)
L4 3 DUP REM L3 (0 DUPLICATES REMOVED)

=> s HLA-A2.1 or HLA-A0201
L5 1283 HLA-A2.1 OR HLA-A0201

=> s l5 and Wilms
L6 11 L5 AND WILMS

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 7 DUP REM L6 (4 DUPLICATES REMOVED)

=> s l7 not l4
L8 5 L7 NOT L4

=> dus l8 1-5 ibib abs kwic
DUS IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> dis l8 1-5 ibib abs kwic

(FILE 'HOME' ENTERED AT 10:25:06 ON 13 MAY 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 10:25:20 ON 13 MAY 2002

L1 2814 S STAUS H?/AU OR GAO L?/AU
L2 3 S L1 AND (GATA OR WT-1)
L3 3 S L1 AND (GATA-1 OR WT-1)
L4 3 DUP REM L3 (0 DUPLICATES REMOVED)

=> s HLA-A2.1 or HLA-A0201
L5 1283 HLA-A2.1 OR HLA-A0201

=> s 15 and Wilms
L6 11 L5 AND WILMS

=> dup rem 16
PROCESSING COMPLETED FOR L6
L7 7 DUP REM L6 (4 DUPLICATES REMOVED)

=> s 17 not 14
L8 5 L7 NOT L4

=> dus 18 1-5 ibib abs kwic
DUS IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> dis 18 1-5 ibib abs kwic

L8 ANSWER 1 OF 5 MEDLINE
ACCESSION NUMBER: 2000197865 MEDLINE
DOCUMENT NUMBER: 20197865 PubMed ID: 10733485
TITLE: Selective elimination of leukemic CD34(+) progenitor cells
by cytotoxic T lymphocytes specific for WT1.
AUTHOR: Gao L; Bellantuono I; Elsasser A; Marley S B; Gordon M Y;
Goldman J M; Stauss H J
CORPORATE SOURCE: Department of Immunology, Imperial School of Medicine,
Hammersmith Hospital, London, UK.
SOURCE: BLOOD, (2000 Apr 1) 95 (7) 2198-203.
JOURNAL CODE: A8G; 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
JOURNAL; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000505
Last Updated on STN: 20000505
Entered Medline: 20000427

AB Hematologic malignancies such as acute and chronic myeloid leukemia are
characterized by the malignant transformation of immature CD34(+) progenitor cells.
Transformation is associated with elevated expression of the Wilms tumor gene encoded transcription factor (WT1). Here we
demonstrate that WT1 can serve as a target for cytotoxic T lymphocytes
(CTL) with exquisite specificity for leukemic progenitor cells.
HLA-A0201-restricted CTL specific for WT1 kill leukemia
cell lines and inhibit colony formation by transformed CD34(+) progenitor
cells isolated from patients with chronic myeloid leukemia (CML), whereas
colony formation by normal CD34(+) progenitor cells is unaffected. Thus,
the tissue-specific transcription factor WT1 is an ideal target for
CTL-mediated purging of leukemic progenitor cells in vitro and for
antigen-specific therapy of leukemia and other WT1-expressing malignancies
in vivo.

AB . . . demonstrate that WT1 can serve as a target for cytotoxic T
lymphocytes (CTL) with exquisite specificity for leukemic progenitor
cells. HLA-A0201-restricted CTL specific for WT1 kill
leukemia cell lines and inhibit colony formation by transformed CD34(+) progenitor cells isolated from. . .

CT
*Antigens, CD34: AN, analysis
Blotting, Western
*Bone Marrow Purging: MT, methods
Child
DNA-Binding Proteins: AN, analysis
*DNA-Binding Proteins: IM, immunology
Genes, Wilms Tumor
Hematopoietic Stem Cells: CH, chemistry
*Hematopoietic Stem Cells: IM, immunology
Hematopoietic Stem Cells: PA, pathology
*Leukemia, Myeloid, . . .

L8 ANSWER 2 OF 5 MEDLINE
ACCESSION NUMBER: 2000130143 MEDLINE
DOCUMENT NUMBER: 20130143 PubMed ID: 10663572
TITLE: Human cytotoxic T-lymphocyte responses specific for
peptides of the wild-type 'Wilms' tumor gene (WT1)
) product.
AUTHOR: Oka Y; Elisseeva O A; Tsuboi A; Ogawa H; Tamaki H; Li H;
Oji Y; Kim E H; Soma T; Asada M; Ueda K; Maruya E; Saji H;
Kishimoto T; Ueda K; Sugiyama H
CORPORATE SOURCE: Department of Molecular Medicine, Osaka University Medical
School, 2-2, Yamada-Oka, Suita City, Osaka 565-0871, Japan.
SOURCE: IMMUNOGENETICS, (2000 Feb) 51 (2) 99-107.
JOURNAL CODE: GI4; 0420404. ISSN: 0093-7711.
PUB. COUNTRY: United States
JOURNAL; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000320
Last Updated on STN: 20000320
Entered Medline: 20000309

AB The product of the Wilms' tumor gene WT1 is a transcription
factor overexpressed not only in leukemic blast cells of almost all
patients with acute myeloid leukemia, acute lymphoid leukemia, and chronic
myeloid leukemia, but also in various types of solid tumor cells. Thus, it
is suggested that the WT1 gene plays an important role in both
leukemogenesis and tumorigenesis. Here we tested the potential of WT1 to
serve as a target for immunotherapy against leukemia and solid tumors.
Four 9-mer WT1 peptides that contain HLA-A2.1
-binding anchor motifs were synthesized. Two of them, Db126 and WH187,
were determined to bind to HLA-A2.1
molecules in a binding assay using transporter associated with antigen

processing-deficient T2 cells. Peripheral blood mononuclear cells from an HLA-A2.1-positive healthy donor were repeatedly sensitized in vitro with T2 cells pulsed with each of these two WT1 peptides, and CD8(+) cytotoxic T lymphocytes (CTLs) that specifically lyse WT1 peptide-pulsed T2 cells in an HLA-A2.1-restricted fashion were induced. The CTLs also exerted specific lysis against WT1-expressing, HLA-A2.1-positive leukemia cells, but not against WT1-expressing, HLA-A2.1-negative leukemia cells, or WT1-nonexpressing, HLA-A2.1-positive B-lymphoblastoid cells. These data provide the first evidence of human CTL responses specific for the WT1 peptides, and provide a rationale for developing WT1 peptide-based adoptive T-cell therapy and vaccination against leukemia and solid tumors.

TI Human cytotoxic T-lymphocyte responses specific for peptides of the wild-type Wilms' tumor gene (WT1) product.

AB The product of the Wilms' tumor gene WT1 is a transcription factor overexpressed not only in leukemic blast cells of almost all patients with acute. . . of WT1 to serve as a target for immunotherapy against leukemia and solid tumors. Four 9-mer WT1 peptides that contain HLA-A2.1-binding anchor motifs were synthesized. Two of them, Db126 and WH187, were determined to bind to HLA-A2.1 molecules in a binding assay using transporter associated with antigen processing-deficient T2 cells. Peripheral blood mononuclear cells from an HLA-A2.1-positive healthy donor were repeatedly sensitized in vitro with T2 cells pulsed with each of these two WT1 peptides, and CD8(+) cytotoxic T lymphocytes (CTLs) that specifically lyse WT1 peptide-pulsed T2 cells in an HLA-A2.1-restricted fashion were induced. The CTLs also exerted specific lysis against WT1-expressing, HLA-A2.1-positive leukemia cells, but not against WT1-expressing, HLA-A2.1-negative leukemia cells, or WT1-nonexpressing, HLA-A2.1-positive B-lymphoblastoid cells. These data provide the first evidence of human CTL responses specific for the WT1 peptides, and provide a . . .

L8 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:210063 CAPLUS
 TITLE: WT1 as a novel target antigen for cancer immunotherapy
 AUTHOR(S): Oka, Y.; Tsuboi, A.; Elisseeva, O. A.; Uda, K.; Sugiyama, H.
 CORPORATE SOURCE: Department of Molecular Medicine, Osaka University Medical School, Suita City, 565-0871, Japan
 SOURCE: Current Cancer Drug Targets (2002), 2(1), 45-54
 CODEN: CCDB9; ISSN: 1568-0096
 PUBLISHER: Bentham Science Publishers Ltd.
 DOCUMENT TYPE: Journal; General Review
 LANGUAGE: English

AB Wild-type Wilms' tumor gene WT1 is expressed at high levels not only in most of acute myelocytic, acute lymphocytic, and chronic myelocytic leukemia, but also in various types of solid tumors including lung cancer. The authors tested the ability of the gene product (WT1) to serve as a target antigen for tumor-specific immunotherapy both in human in vitro system and mouse in vivo system. In the latter, the authors can evaluate the efficacy and the side effects of WT1 vaccination in vivo. In the human in vitro system, two WT1 peptides that contain HLA-A2.1 binding anchor motifs were detd. to bind to HLA-A2.1 mols. Peripheral blood mononuclear cells (PBMC) from an HLA-A2.1-pos. donor were repeatedly stimulated in vitro with TAP-deficient T2 cells pulsed with each of these two peptides, and CD8-pos. cytotoxic T lymphocytes (CTLs) that specifically lyse WT1-expressing, HLA-A2.1-pos. tumor cells were induced. Other groups also have succeeded in generating CTLs which specifically lyse WT1-expressing leukemia cells, and which do not inhibit colony-formation of normal hematopoietic cells that express WT1 at physiol. levels. In the mouse in vivo system, immunization of C57BL/6 mice with one WT1 peptide with relatively high binding affinity for H-2Db mols., which contain H-2Db binding anchor motifs, induced CTLs, which specifically lysed WT1-expressing tumor cells in an H-2Db-restricted manner. Furthermore, mice immunized with the WT1 peptide (peptide vaccination) or WT1 cDNA (DNA vaccination) rejected challenges by WT1-expressing tumor cells and survived with no signs of autoaggression to WT1-expressing normal organs by the induced CTLs. The WT1 protein has been identified as a novel tumor antigen and recent investigations provide a rationale for developing WT1-based adoptive T cell therapy and vaccination against various kinds of malignant neoplasms.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Wild-type Wilms' tumor gene WT1 is expressed at high levels not only in most of acute myelocytic, acute lymphocytic, and chronic myelocytic leukemia, but also in various types of solid tumors including lung cancer. The authors tested the ability of the gene product (WT1) to serve as a target antigen for tumor-specific immunotherapy both in human in vitro system and mouse in vivo system. In the latter, the authors can evaluate the efficacy and the side effects of WT1 vaccination in vivo. In the human in vitro system, two WT1 peptides that contain HLA-A2.1 binding anchor motifs were detd. to bind to HLA-A2.1 mols. Peripheral blood mononuclear cells (PBMC) from an HLA-A2.1-pos. donor were repeatedly stimulated in vitro with TAP-deficient T2 cells pulsed with each of these two peptides, and CD8-pos. cytotoxic T lymphocytes (CTLs) that specifically lyse WT1-expressing, HLA-A2.1-pos. tumor cells were induced. Other groups also have succeeded in generating CTLs which specifically lyse WT1-expressing leukemia cells, and which do not inhibit colony-formation of normal hematopoietic cells that express WT1 at physiol. levels. In the mouse in vivo system, immunization of C57BL/6 mice with one WT1 peptide with relatively high binding affinity for H-2Db mols., which contain H-2Db binding anchor motifs, induced CTLs, which specifically lysed WT1-expressing tumor cells in an H-2Db-restricted manner. Furthermore, mice immunized with the WT1 peptide (peptide vaccination) or WT1 cDNA (DNA vaccination) rejected challenges by WT1-expressing tumor cells and survived with no signs of autoaggression to WT1-expressing normal organs by the induced CTLs. The WT1 protein has been identified as a novel tumor antigen and recent investigations provide a rationale for developing WT1-based adoptive T cell therapy and vaccination against various kinds of malignant neoplasms.

IT Transcription factors
 RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (WT1 (Wilms' tumor suppressor 1); for cancer immunotherapy)

L8 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2001:904730 CAPLUS
 DOCUMENT NUMBER: 136:36345

TITLE: Artificial antigen presenting cells and methods of use thereof
 INVENTOR(S): Sadelain, Michel; Latouche, Jean-Baptiste
 PATENT ASSIGNEE(S): Memorial Sloan-Kettering Cancer Center, USA
 SOURCE: PCT Int. Appl., 75 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001094944	A2	20011213	WO 2001-US17981	20010601

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-209157P P 20000602

AB The invention provides an artificial antigen presenting cell (AAPC) comprising a eukaryotic cell expressing an antigen presenting complex comprising a human leukocyte antigen (HLA) mol. of a single type, at least one exogenous accessory mol. and at least one exogenous T cell-specific epitope. Methods of use for activation of T lymphocytes are also provided. Fibroblasts were retrovirally transduced with an HLA-peptide complex and the accessory mols. B7.1, ICAM-1, and LFA-3. These AAPCs elicit strong stimulation and expansion of CTLs, and may be used in therapy for a no. of diseases.

IT Histocompatibility antigens
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (HLA-A2.1; artificial antigen presenting cells expressing HLA class I, peptide, and accessory mols. for activation of cytotoxic T cells and adoptive immunotherapy)

IT Antitumor agents
 (Wilms' tumor; artificial antigen presenting cells expressing HLA class I, peptide, and accessory mols. for activation of cytotoxic T cells and adoptive immunotherapy)

IT Kidney, neoplasm
 (Wilms', inhibitors; artificial antigen presenting cells expressing HLA class I, peptide, and accessory mols. for activation of cytotoxic T cells and adoptive immunotherapy)

L8 ANSWER 5 OF 5 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
 ACCESSION NUMBER: 2002:129944 BIOSIS
 DOCUMENT NUMBER: PREV200200129944
 TITLE: Identification of a novel WT1 HLA A*0201-restricted CTL epitope using whole gene in vitro priming.
 AUTHOR(S): Smithgall, Molly (1); Misher, Linda (1); Spies, Greg (1); Cheever, Martin A. (1); Gaiger, Alexander (1)
 CORPORATE SOURCE: (1) Immunology, Corixa, Seattle, WA USA
 SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 121a. <http://www.bloodjournal.org/>. print.
 Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001
 ISSN: 0006-4971.
 DOCUMENT TYPE: Conference
 LANGUAGE: English

AB The Wilms tumor (WT1) protein is a self-protein recently identified as a candidate antigen for leukemia vaccine and T-cell therapy. The current study assessed the feasibility of generating WT1 specific T-cell responses using whole gene in vitro priming. The advantages of whole gene in vitro priming are 1) the entire spectrum of epitopes of a given protein is present, 2) selection and presentation of these naturally processed peptides are done by the antigen presenting cell. Monocyte derived dendritic cells (DC) of HLA A0201 positive normal donors were infected with replication-deficient recombinant adenovirus (Adeno) or vaccinia virus (Vac) expressing full length WT1. CD8+ T-cell cultures were restimulated every 7-10 days, alternating Adeno/WT1 infected autologous DC with Vac/WT1 infected DC. T cell responses were evaluated by measuring levels of interferon-gamma secretion by ELISPOT analysis in response to WT1 expressing target cells. After 4 stimulation cycles, CD8+ T cell lines that specifically, recognized WT1 transduced autologous fibroblasts, but not control transduced fibroblasts, were identified and cloned. HLA A2 restriction of the clonal T-cells was documented by 1) antibody blocking experiments and 2) recognition of WT1 transduced fibroblasts derived from a second donor, who shares only the HLA A2 allele with the original donor. Recognition of leukemia cells "naturally" overexpressing WT1 by the CTL clone was shown by recognition of HLA A2 transduced WT1 overexpressing cell line K562 but not of HLA class I negative control transduced K562 cells. Using truncated WT1 retroviral constructs to transduce autologous fibroblasts the WT1 epitope was localized to the first 92 N-terminal aminoacids of the WT1 protein. Using overlapping WT1 peptides the epitope was further localized to aa37-47. All 9mer WT1 peptides within this region were synthesized. The CD8+ clone specifically recognized the 9mer VLDPAPPGA (aa37-45), demonstrating that this WT1 peptide is a naturally processed HLA A0201 restricted epitope. The ability to generate WT1 specific CD8+ T-cell clones and clone their T-cell receptor might allow treatment of malignancies associated with WT1 overexpression using genetically engineered T-cells. These data provide further validation of WT1 as a leukemia vaccine and T-cell therapy candidate.

AB The Wilms tumor (WT1) protein is a self-protein recently identified as a candidate antigen for leukemia vaccine and T-cell therapy. The current . . . and presentation of these naturally processed peptides are done by the antigen presenting cell. Monocyte derived dendritic cells (DC) of HLA A0201 positive normal donors were infected with replication-deficient recombinant adenovirus (Adeno) or vaccinia virus (Vac) expressing full length WT1. CD8+ T-cell. . . were synthesized. The CD8+ clone specifically recognized the 9mer VLDPAPPGA (aa37-45), demonstrating that this WT1 peptide is a naturally processed HLA A0201 restricted epitope. The ability to generate WT1 specific CD8+ T-cell clones and clone their T-cell receptor might allow treatment of. . .

IT . . . antigen presenting cell; immune system; dendritic cell; immune system; monocyte; blood and lymphatics, immune system

IT Chemicals & Biochemicals
HLA-A-0201; Wilms tumor protein [WT1]: expression
GEN HLA A2 gene: allele; WT1 gene [Wilms tumor gene]: epitopes

=> s rmfpnapyl
L9 1 RMFPNAPYL

=> dis l9 ibib abs

L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:314730 CAPLUS
DOCUMENT NUMBER: 132:333396
TITLE: Immunotherapy of cancer using epitopes of WT-1 and
GATA-1 transcription factors
INVENTOR(S): Stauss, Hans Josef; Gao, Liqun
PATENT ASSIGNEE(S): Imperial College Innovations Limited, UK
SOURCE: PCT Int. Appl., 93 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000026249	A1	20000511	WO 1999-GB3572	19991102
W:	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LJ, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 9964797	A1	20000522	AU 1999-64797	19991102
EP 1127068	A1	20010829	EP 1999-952682	19991102
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO			

PRIORITY APPLN. INFO.: GB 1998-23897 A 19981102
WO 1999-GB3572 W 19991102

AB The authors disclose that the peptides **RMFPNAPYL** or **CMTWNQMNL** are epitopes for cytotoxic T-cells recognizing WT-1 in an HLA-A2-restricted manner. In addn. the peptide is **HLMPFPGPLL** is a CTL epitope of human GATA-1 transcription factor. The peptides, and polynucleotides encoding them, may be useful as cancer vaccines.
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> end

ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF

LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
47.77	47.98

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-2.48	-2.48

CA SUBSCRIBER PRICE

STN INTERNATIONAL LOGOFF AT 10:30:28 ON 13 MAY 2002